

# Accelerated atrioventricular conduction after myocardial infarction

## *A study using His bundle electrograms*

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*His bundle recordings obtained in a patient with progressive shortening of the PR interval observed during and after acute diaphragmatic myocardial infarction showed abbreviated AH and HV intervals. Though atrial pacing at rapid rates produced prolongation of the AH interval, this was not as great as is seen in normal subjects and the abbreviated HV interval remained unchanged. The results of His bundle electrograms during atrial pacing on another patient with an old inferior myocardial infarction and intermittent Wolff-Parkinson-White syndrome were similar to the response of normal subjects, but the Wolff-Parkinson-White configuration was only seen with premature supraventricular beats.*

*Possible mechanisms are discussed, and it is suggested that the most likely is infarction of the AV nodal tissue responsible for the normal nodal delay.*

Accelerated atrioventricular (AV) conduction associated with myocardial ischaemia is rare, though all grades of AV block are commonplace. Accelerated AV conduction manifesting as Wolff-Parkinson-White syndrome (anomalous AV excitation) has been reported to occur after acute carbon monoxide poisoning (Seling, 1966), during acute articular rheumatism (Ougier, Page, and Marc, 1964), in myocarditis, cardiomyopathy, and digitalis toxicity (Ohnell, 1944; Vakil, 1955; Mathur, Wahal and Seth, 1969), and in some congenital heart diseases. There have been occasional reports of Wolff-Parkinson-White syndrome occurring with acute myocardial infarction (Apostolov, 1964; Forin and Tammaro, 1969; Angelino, Mina, and Gallo, 1964).

Two patients with accelerated AV conduction associated with acute ischaemic heart disease have recently been observed and studies performed to elucidate the mechanisms by which this may occur.

### Methods

Both patients were studied in the postabsorptive state and were premedicated with 10 mg diazepam intramuscularly one hour before cardiac catheterization. The procedure was explained to the patients in detail and the necessary consent was obtained. Under 1 per cent xylocaine local anaesthesia, a bipolar (10 mm) 6 Fr. electrode catheter

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was introduced into the right or left femoral vein percutaneously. The catheter tip was positioned using fluoroscopic control across the tricuspid ring, as described by Scherlag *et al.* (1969) and Damato and Lau (1970).

His bundle potentials were recorded over a frequency range of 40-500 Hz on a Cambridge six-channel photographic recorder at a paper speed of 100 mm/sec.

A second 5 Fr. bipolar electrode catheter was passed percutaneously from the right subclavian vein and positioned at the site of the sinoatrial node, for pacing the right atrium from an external battery-powered pacemaker up to a rate of 170/min.

Atrium to His bundle (AH) times were measured from the P wave of a simultaneous standard lead III, and His to ventricle (HV) times from the intracardiac recording.

### Case reports

#### Case 1

A 59-year-old man with a two-year history of palpitation and a three-month history of dizzy attacks, was admitted to hospital in March 1972, with crushing chest pain which started suddenly while he was gardening. The pain radiated to his left arm without dizziness or shortness of breath. Physical examination revealed a small man of normal nutrition. There was no clinical evidence of hypercholesterolaemia. His pulse was 54/min when admitted and later rose to 73/min spontaneously. His blood pressure was 120/90 mmHg. There were no signs of heart failure and his peripheral pulses were normal.

The remainder of the physical examination was within normal limits. His electrocardiogram showed sinus rhythm with a PR interval of 0.18 sec and an acute inferior (diaphragmatic) myocardial infarction, and he was admitted to the coronary care unit for treatment. His progress was uneventful and there were no compli-

cations. No drugs were administered and his serum electrolytes were within normal limits. Serial electrocardiograms showed evolutionary changes and a decreasing PR interval, from 0.18 to 0.12 sec, from the first day of admission onwards (Fig. 1). The shortened PR interval has remained constant ever since.

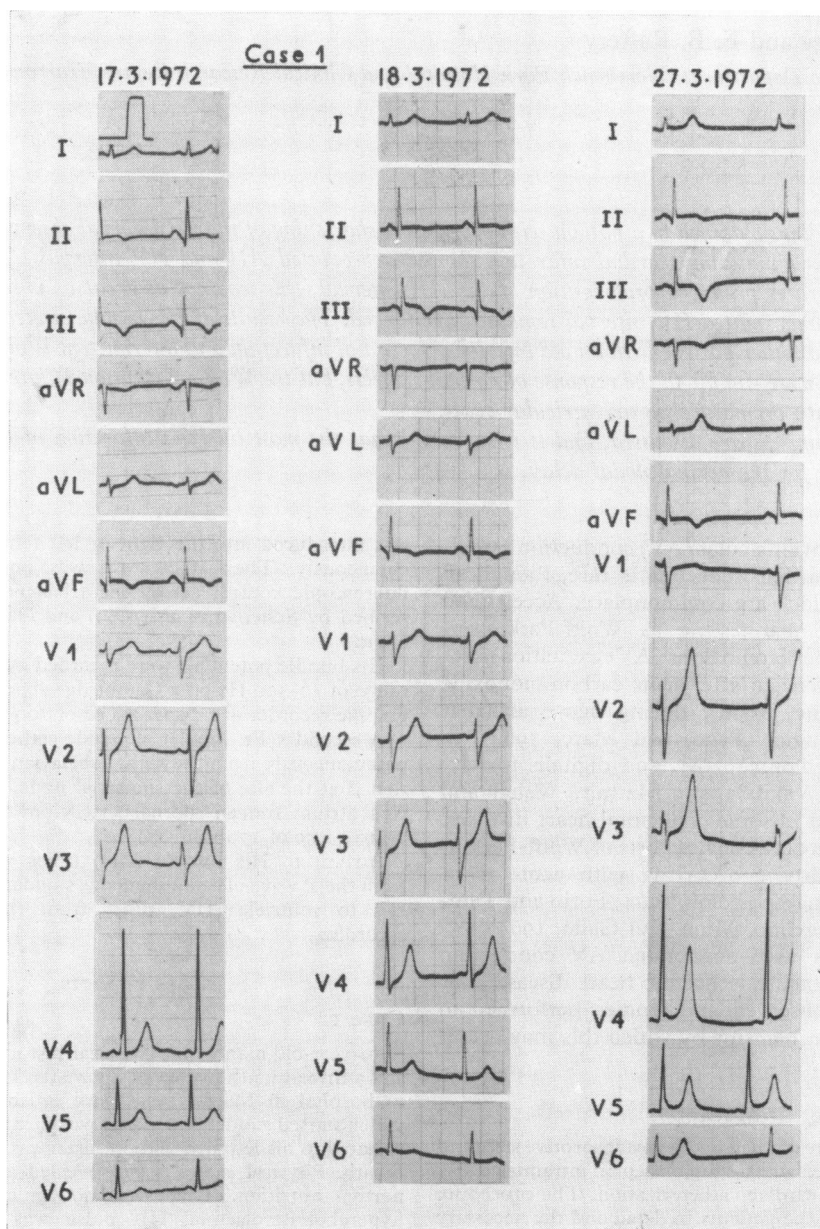


FIG. 1 Successive electrocardiograms from Case 1 showing the evolution of a diaphragmatic infarction with shortening of the PR interval from 0.18 sec to 0.12 sec.

TABLE Measured electrogram intervals during atrial pacing

Heart rate (beats/min)	Case 1		Case 2	
	AH* (msec)	HV† (msec)	AH* (msec)	HV† (msec)
68	100	24	—	—
100	120	26	150	48
110	148	24	180	48
120	152	22	210	48
130	180	22	230	48
140	184	22	260	50
150	200	24	290	50
160	200	22	300	48
170	200	22	300	48

\* 'Normal' value, 120–200 msec.

† 'Normal' value, 35–55 msec.

**Electrophysiological study and results** The components of the His bundle electrogram during sinus rhythm and with atrial pacing up to 170/min are summarized in the Table. Representative tracings during pacing at 140/min and 150/min are shown in Fig. 2. The AH interval was abbreviated at rest and with increasing pacing rates it was prolonged, but not to the degree usually described for normal subjects (300 msec at 160/min), but the HV interval, which was abbreviated to begin with, remained the same. This suggested a partial atrioventricular (AV) nodal bypass which responded to increasing atrial rates in a normal fashion, and rapidly-conducting fibres in the His bundle revealed as a consequence of ischaemia of the AV node.

### Case 2

A 53-year-old man was admitted to the hospital complaining of orthopnoea, paroxysmal nocturnal dyspnoea,

Case 1 Lead III

Rate = 140/min

Rate = 150/min

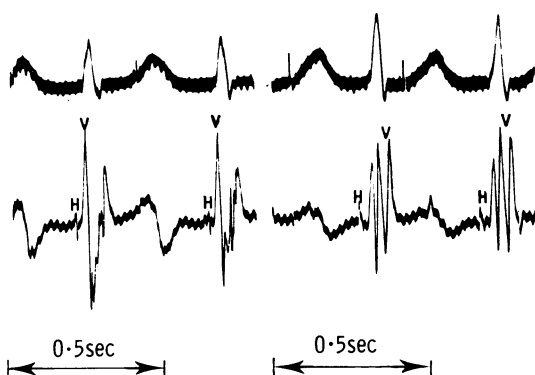


FIG. 2 His electrograms from Case 1 during atrial pacing. Note the short AH and HV intervals. H, His potential; V, ventricular potential.

and increasing angina. He had a past history of antero-inferior myocardial infarction in 1966 and had been out of work because of his cardiac disability. Physical examination disclosed a blood pressure of 140/80 mmHg, and a pulse rate of 100/min, with occasional ectopic beats. There was no evidence of congestive heart failure but his heart was enlarged with a loud third heart sound and an intermittent fourth heart sound. His lungs were clear. The rest of the physical examination was within normal limits. An electrocardiogram showed old anterior and inferior myocardial infarction and occasional premature atrial contractions with the Wolff-Parkinson-White type of aberrant intraventricular conduction (Fig. 3). He was investigated with right and left heart catheterization including coronary arteriograms. Because of the intermittent Wolff-Parkinson-White complexes on the electrocardiogram, he also had a His bundle electrogram. During his hospital stay his serum electrolytes and serum enzymes were within normal limits. The coronary arteriogram showed complete occlusion of right coronary artery with some collaterals from the circumflex branch. A left ventriculogram showed poor contraction, particularly of the diaphragmatic surface.

**Electrophysiological study** The AH interval and HV intervals were measured before and after atrial pacing (Table), and followed the pattern seen in normal subjects. The frequency of premature atrial contractions diminished during atrial pacing but they still occurred sporadically; tracings showed the His spike buried within the ventricular complex (Fig. 4). This suggested that anomalous AV conduction was occurring with premature supraventricular beats, but there was no evidence of rapid conduction below the AV node.

### Discussion

Anatomical studies summarized by James (1966) and James and Sherf (1968) suggest that the sinoatrial node is connected to the atrioventricular (AV) node by means of three specialized tracts in the atrial muscle (Fig. 5). The sinoatrial impulse is conducted rapidly in these specialized tracts, particularly the anterior and middle tracts, and reaches the crest of the AV node, where a delay of about 5 msec occurs before the node is depolarized. The mechanism of this delay is obscure, but it is thought to occur at the junction of transitional cells and P cells. The posterior tract is larger than the other two and is activated at a later stage. Its terminal fibres make connexions with the bundle of His below the AV node and have the potential of bypassing the nodal delay (Fig. 4), but the impulses which travel by this route are usually blocked before they can be conducted. Sherf and James (1969) suggest that functional disturbances in the SA node can produce a situation in which the posterior tract is activated sooner than the other tracts. The net result of this would be a short PR interval with a normal QRS

complex, or a Wolff-Parkinson-White syndrome, depending on whether Mahaim fibres were activated or not.

The studies performed on the first of our patients suggest accelerated conduction both above and below the AV node. In this respect the findings differ from those described in Lown-Ganong-Levine syndrome (Smithen and Krikler, 1972), but are similar to the findings reported in a single case of Lown-Ganong-Levine syndrome by Mandel, Danzig, and Hayakawa (1971).

These findings are most readily explained by postulating that the area of the AV node responsible for the normal delay process has been destroyed, and that rapidly-conducting pathways in the bundle of His such as those described by Mendez and Moe (1966) have been activated. Proof of this would be provided by His electrograms during retrograde conduction of artificially-induced right ventricular ectopic beats, but unfortunately it was not possible to make this observation on this patient.

The studies performed on the second patient were

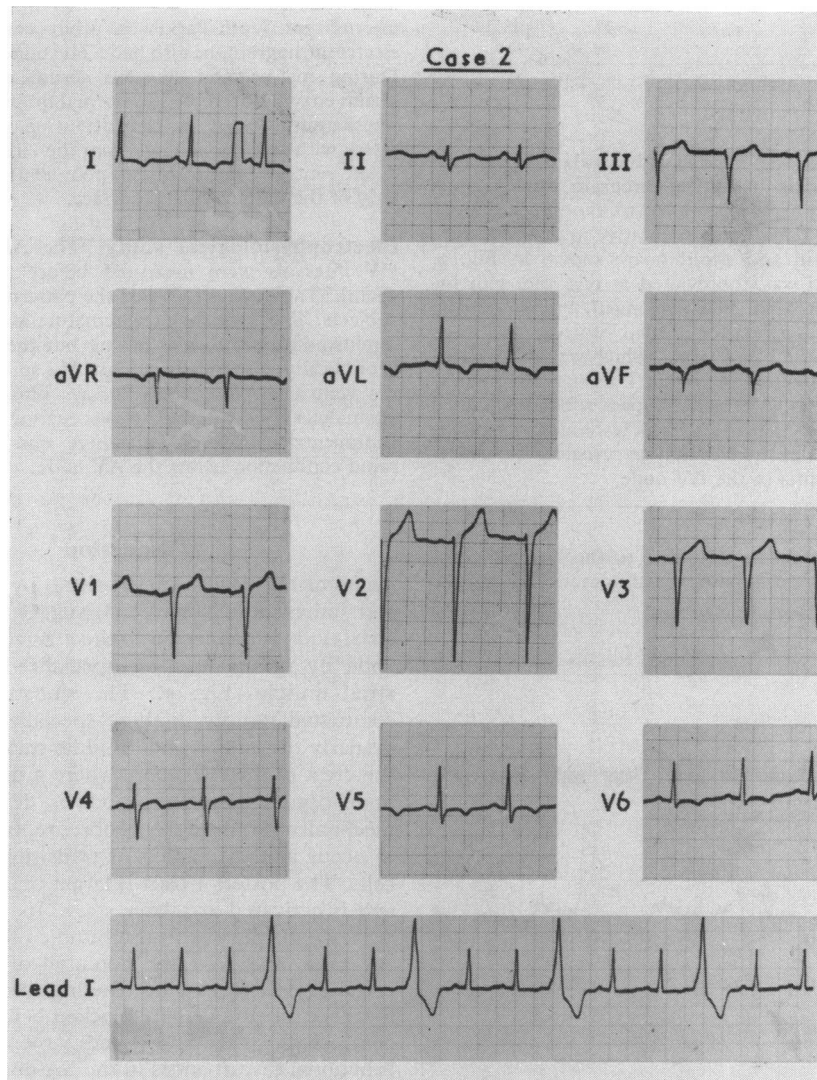


FIG. 3 *Electrocardiogram from Case 2. Note slightly premature ectopic beats in the rhythm strip with the characteristic appearance of Wolff-Parkinson-White syndrome.*

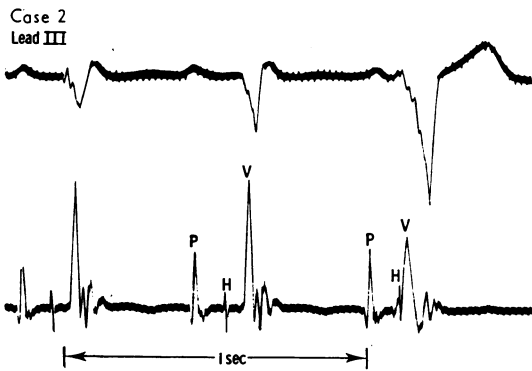


FIG. 4 His electrogram from Case 2. The third beat, which is not premature, shows the Wolff-Parkinson-White configuration with the His impulse distorting the V complex. P, atrial potential; H, His potential; V, ventricular potential.

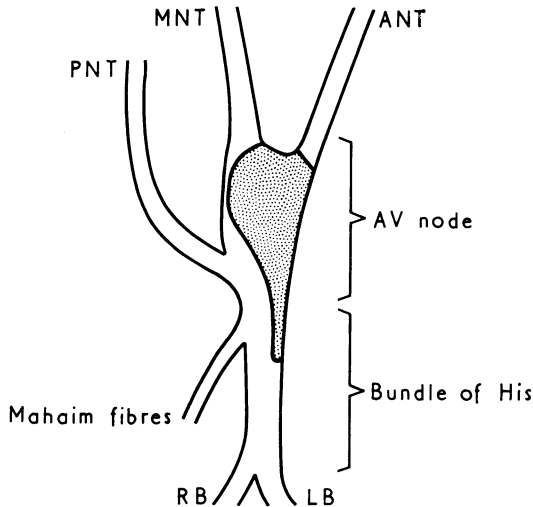


FIG. 5 Diagram to illustrate the conduction pathways through the AV node. Hatched area represents hypothetical ischaemia and nonfunctional area of the node. ANT, anterior nodal tract; MNT, middle nodal tract; PNT, posterior nodal tract; LB, left bundle; RB, right bundle.

amenable to much simpler interpretation, and suggest early activation of the posterior interatrial tract with ectopic beats, though intermittent activation of a Kent bundle or a Mahaim bundle cannot be excluded.

The right coronary artery always provides the blood supply to the AV node, and in 55 per cent of cases it supplies the sinoatrial node as well. It is not surprising that infarction or ischaemia involving this vessel commonly produces significant alterations in atrioventricular conduction, but these alterations

usually take the form of heart block and it is not common to find accelerated conduction. The mechanisms of accelerated conduction in these circumstances must remain speculative, but the most likely explanation is that partial infarction or ischaemia of the AV node leaves a very small remaining functional area which is unable to interpose the normal nodal delay. However, our observations suggest that this explanation alone is not enough, and in order to explain the rapid conduction both above and below the AV node, it is necessary to postulate a system of rapidly-conducting fibres in the conducting system, whose presence has been unmasked by the nodal infarction. Evidence that such systems may exist has been produced by Mendez and Moe (1966) and Prinzmetal *et al.* (1952).

The eventual outcome of accelerated atrioventricular conduction after myocardial infarction are not known. Whether resolution of the acute phase will restore the normal conduction pattern or not remains to be seen, but it seems likely that progressive ischaemia will eventually result in heart block in these individuals.

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